Vol. IV. January, 1938 No. I.

DISEASES OF THE CHEST

Official Organ of the Amer. College of Chest Physicians
Editorial offices 1018 Mills Building, El Paso, Texas
Business Address Myrtle at Virginia, El Paso, Texas
MEMBER: ASSOCIATED EDITORS OF TUBERCULOSIS PUBLICATIONS

(A MONTHLY PUBLICATION)

Subscription: United States and Canada $2.00 per year. Other countries $2.50 per year.
Entered as second-class matter August 18, 1936, at the post office at El Paso, Texas, under the Act of August 24, 1912.

Editorial Comment

THE SEASON'S GREETINGS WE have started the New Year with a number of changes in DISEASES OF THE CHEST. We hope that you will take notice of our new cover, the change of format in arranging papers, the new style of type, the heavier paper, and the increased width of our journal. We express our thanks to the Fellows who have given us suggestions for the improvement of the journal and we at all times solicit your further comments.

It is the desire of the Editorial Board to maintain the same progressive attitude for DISEASES OF THE CHEST, which was inaugurated at the very beginning of the journal. We will endeavor to express through the columns of the journal, the high purposes and aims of the American College of Chest Physicians. Here, again, we solicit the co-operation of the Fellows of the American College of Chest Physicians and the readers of DISEASES OF THE CHEST.

We will continue to publish practical papers by competent authors stressing the diagnosis and treatment of chest diseases.

The journal is so designed that it will interest the physician in the general practice of medicine as well as the chest specialist.

The Editorial Board of DISEASES OF THE CHEST extends the Season's Greetings to all of its readers and to the Fellows of the American College of Chest Physicians. F. W. B.

PHILADELPHIA PLAN IS SPREADING FROM North, South, and West, come inquiries concerning the Philadelphia Plan of a nation - wide organization of Tuberculosis - Minded Physicians within Organized Medicine. From several States have come assurances from such men that they are actively organizing Tuberculosis Committees in their County Medical Societies and are planning to organize a Committee in their State Medical Societies as soon as a meeting takes place.

Fortunate indeed (and few), are the tuberculosis patients in communities where the sanatorium service, case finding, nursing, and rehabilitation, are without defect. In those communities, a Committee should be formed to disseminate information about such perfection, to change such backward situations as exist elsewhere.

Physicians in organized medicine alone are capable of organized effort to bring treatment to as high a standard as it is in some spots, and should be everywhere.

Will you write and tell us the most glaring defect in your Tuberculosis System? We want your influence and help with ours (insufficient medical personnel and Pneumothorax Operators who are untrained). Our Tuberculosis Nurse Training is unsatisfactory. Is yours? Can you advise us? — Frank Walton Burge, 1930 Chestnut Street, Philadelphia, Pa.

NOT OVER FIFTY TO ONE

FIFTY patients to one doctor is the maximum compatible with modern study and treatment necessary in the Tuberculosis Sanatorium today. Some sanatoriums have only 30 patients to 1 doctor.

If your sanatorium has too many patients leaving "against advice" do not try to explain it on grounds of the inferiority of the patient; it is always due to some inferiority in the sanatorium.

It is impossible for an average doctor to be keenly interested in the individual case when he is unable to keep up with the bare necessities.

The authorities of the World are agreed that every pneumothorax case should be fluoroscoped before every refill. When a patient accustomed to such procedure goes to a sanatorium where he receives refill after refill without fluoroscopic study, do you think he can have confidence in that sanatorium's treatment?

Yet in many institutions for treatment of tuberculosis the doctor cannot take time to fluoroscope the hundreds of cases under his care before each refill; nor can he study each case as it needs study today.

So, for the sake of the poor neglected patient in the under-staffed sanatorium, let us unite in demanding "Not over 50 to 1".

F. W. B.

A letter received from a physician in Florida.

AN EDITOR SHOULD NOT MAKE AMBIGUOUS STATEMENTS

"FOR two years or thereabout, I have not missed reading every page of your journal, and I have always reveled in it, but I must admit the November issue stunned me. If I have been able to sense your articles the one continuous thread above all others has been "Find Tuberculosis Early That It May Be Gotten At In Time. Now, I read on page six of the present issue some one begrudging what little an interne learns from examining the host of patients that he is serving day after day with no other remuneration than what he learns from them. Will the author kindly tell me just when and how does he expect the interne to be able to recognize tuberculosis if he is not able to get a little from interning? I thought that was the price charity patients paid for the work that the interne staff does for them free of charge. I just cannot see how anyone would ever learn any medicine if he is not to even examine anyone. Will he wait till he gets out in general practice and try to learn on the ones who are paying him money. Certainly I would not interne at a hospital that refused me the right to examine the patients for whom I worked day after day. Interne life is hard and the poor devil certainly rates a little chance to improve his knowledge since he does not even get a few dollars for smokes after serving years in school. Certainly I would not abuse the patient, but I maintain "that little knowledge" must be attained before he can secure "that big knowledge". Where is he to start examining patients? And when?

Very sincerely yours, M. D."

November 2, 1937

"————, Florida,

"Dear Doctor . . . . . :

"Your letter of November 2nd, 1937, is indeed gratefully received because it teaches me again how we must never take anything for granted and lack completeness in any article.

"I have been campaigning for sometime against untrained pneumothorax operators. In some of our hospitals, internes on a two-months rotating interneship do practically all of the pneumothorax work in the hospital. By the time they are slightly trained, they move on. In the editorial to which you referred in your letter, no objection whatever was intended to the interne acquiring all of the skill in diagnosis of which he is capable. The objection was purely to his doing operative procedures for which he is improperly trained.

"We maintain that the only people who should be trained to do pneumothorax are men who intend making chest work a specialty.

"Thank you for your kindness and I hope you will help in the future with any mistakes we may make.

"Very cordially yours, F. W. B."

November 11, 1937

THE CAVITY

IN the parade of pathological changes taking place in the lung structure along the whole tuberculosis front, the most formidable is the open cavity. It is the chief source of spread of the disease both within and without the body. Could only an Aladdin come among us and stroke his magic lamp, and forthwith command that all existing cavities immediately close; and that no other case progress to cavitation! With this state of affairs, the propagation of tuberculosis would practically cease. The disease having no victims about which to entwine its life-blood sucking tentacles, would shortly wither and die. We can invoke no Aladdin and his lamp; but by the persevering, rigid, diligent and skillful application of many measures at our disposal, we can anticipate this eventuation. These measures include among others such vital ones as examination of contacts; mass tuberculin testing with x-ray examination of the postive reactors; a complete examination of all members of a household where there is a postive reactor, particularly one in the pre —or early school age; the segregation of all advanced cases; and the attempt by some form of collapse therapy to convert postive suptum cases to negative cases — which means closing their cavities.

Every case under treatment should be closely followed at all times, with frequent sputum and periodic x-ray examinations. An amelioration or disappearance of symptoms, a sense of well being and a gain in weight may be misleading. These may occur during a time when the disease process within the lungs may be undergoing a tragic spread. The most satisfactory index to the progress of a patient is the sputum and x-ray examination. A negative sputum and film showing no open cavity indicate accurately that things are going well; but conversely, an open cavity and a postive sputum portend trouble ahead.

Recent statistics reveal that in 80% of cases when the diagnosis of tuberculosis is first made, cavities are demonstrable; and they are likewise present in 85% of all admissions to the sanatorium. Surely this would tend to prove that we are yet far removed from our goal. Where possible, cavities must be closed. Where impossible — and with the rapid advances in collapse therapy this group is becoming progressively smaller — these cases should be segregated. An open cavity is always a potential, and usually a real, menace. It is a menace to the individual who harbors one, in that it is the frequent cause of a bronchogenic spread to other parts of the lung or even to the opposite lung. It is a menace to the public at large in that it is the source of innumerable tubercle bacilli discharged into the outside world to infect other innocent and unsuspecting victims.

When there are large numbers of tubercle bacilli in the sputum, it is presumptive evidence of the presence of larger or smaller cavities. The most reliable means for their detection is the x-ray; but they are not always revealed on the film even when they may be proved to exist. By the use of Tomography (x-ray exposures made at varying depths within the lungs) cavities can be shown which were not disclosed by the usual procedures. The persistence of metallic, consonating or large moist rales over an area in the upper lobe, with or without amphoric or cavernous breathing and the classical post tussic suction sound, is reliable evidence of excavation. A postive sputum under these conditions is practically confirmatory.

It may be well to again emphasize that an appropriate slogan in our campaign against tuberculosis would be CLOSE THAT CAVITY.

C. H. H.

NEW YEAR'S RESOLUTIONS

Resolved: That we will continue to look for the early case of tuberculosis.

Resolved: That we will examine and tuberculin test all contacts, and that we will x-ray all postive reactors.

Resolved: That we will institute early collapse of diseased lungs.

Resolved: That we will isolate all open cases of tuberculosis.

Resolved: That we will support the educational program of the American College of Chest Physicians, and the National Tuberculosis Association.

Resolved: That we will subscribe to and support the journal, DISEASES OF THE CHEST.

The Psychology of The Tuberculous Patient

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WHEN the practical homespun psychology of the family physician yielded to the pressure of the newly developing scientific approach to medicine, the community suffered a great loss. While prescientific medicine had many weaknesses it had always been a real staff on which suffering humanity could lean in times of need.

The change from giving most attention to relieving the patient's symptoms and assuring him and the family that he would soon be more comfortable, be relieved of pain, or be out of danger, to that of making a diagnosis; was a shift from making the patient the object of greatest concern to that of substituting interest in the disease for interest in the patient. This was an instance in which, while training the backward horse of the team we neglected the other and let him become incompetent in comparison.

In order to understand the psychology of the tuberculous patient we must conceive of man in his dual relationship of a physical machine housing a psychical being. While all human bodies have essentially the same parts, and while the psychical or emotional side of man, as well as his physical being, shows similar characteristics in all human beings, yet our study of both the physical and psychical must be directed to the individual. While certain characteristics are found in common, those which are of greatest importance to man in health or disease are those which are peculiar to the individual in review.

We wholly miss the essence of the disease, tuberculosis, unless we proceed from the standpoint that the physical mechanism only houses the individual who is ill. If the tuberculous patient were not a thinking, wishing, hoping individual, subject to the effects of joy, happiness, sorrow, disappointment, discouragement and despair; and if these states did not react favorably or unfavorably upon his progress toward recovery, the problems of the physician who guides the patient would be comparatively easy.

In the first place, let us inquire what are we trying to accomplish in the treatment of tuberculosis? Are we trying to heal a lung or joint or bone just that they may be restored in their integrity? Far from it, we are attempting to restore these members to a state of integrity so that the being housed in the injured body may take his place in life capable of enjoying and accomplishing; otherwise our efforts are misdirected. This is what gives us the greatest problems in the therapy of the tuberculous. It is better to say the treatment of the tuberculous patient rather than the treatment of tuberculosis, for this emphasizes the patient rather than the disease.

The reaction toward disease is both physical and psychical. These two aspects of man are so closely bound and their reactions are so nicely integrated that reaction in one affects the other, the nature of the individual and the amount of the reaction depending primarily on that basic condition which may be taken as his usual or predominant status, and secondarily, upon modifying factors.

For our purpose it is fruitless to discuss whether disturbances in the physical machine or in the psychical being are primary in the makeup of a given individual, but we do know that the composite physical and psychical being shows many variants and that reaction to disease or to any other stimulus is individual.

Just as we know man's personality may be altered according to the state of his physical body, so can the functions and likewise the structure of his body be changed by varying psychological states.

The general effect of tuberculous disease on the body and likewise upon the psychical aspects of the individual will vary according to its extent and severity. In general, its primary effect is that of injuring and destroying structures locally and of altering function throughout the body; but a disturbed function causes worry, fear, anxiety, and depression; causes the patient to be cognizant of body functions, which should be carried on unnoticed, and has a tendency to

cause a centering of his attention upon himself. This may lead to self-pity, fear, pessimism, and defeat, all of which again react through the nervous and endocrine systems of the patient to further stimulate changes in function which may already be disturbed or to create new departures from normal.

The effect of a wrong diagnosis of tuberculosis, or of the patient suspecting it, is often apparent in an increase in nervousness, insomnia, feelings of tiredness, lack of vigor, loss of appetite, disturbed digestion, loss of weight, increased heart action and shortness of breath. When, after an adequate examination, the patient is told that active tuberculosis does not exist these symptoms will often disappear like magic. They are psychical in origin and are relieved by changing the psychology of fear and despondency to one of confidence and happiness.

When a definite diagnosis is made, however, then there is a mixed psychologic condition brought about based on the necessity of facing the fact of a serious illness which removes one from his accustomed work, separates him from his domestic, social, and business activities and carries with it a certain amount of uncertainty for the future. This is mitigated somewhat by the physician's statement of the hopefulness connected with the proper treatment and the immediate institution of the same. At best, however, there is always some doubt, unhappiness, disappointment, worry, and depression connected with the illness and the sacrifices that must be made. These gradually may be displaced by proper suggestion. To appreciate the tuberculous patient's problem one must think of the number of ways in which one facing the fact of having active tuberculosis must reorganize his life and activities as he plans to face the future.

A physician, to meet such situations, must be more than a physician. Above all, he must be a humanitarian. He must be able to appreciate others' problems and be sympathetic toward them and their solution. He must not only be a counselor but a wise counselor. He must understand that he has not a tuberculous lesion alone to heal but a human being to restore to healthful thinking and living, so that he may take his place in enjoying life and producing to the extent of his ability; adding his part to the sum total of the world's accomplishment.

There is a tendency for a chronic illness like tuberculosis which is attended by toxic absorption, and which requires much rest and often divorcement from business activity and social responsibility for months and sometimes many months, to make patients dependent and childlike in their psychology, to produce in them such a psychic trauma that they lose confidence and shrink from facing life's problems which await them on recovering. They fear the future at times more than they fear the disease. The prevention of this state of dependence can be best accomplished. by early diagnosis and immediate application of adequate treatment. Such a program should restore ninety per cent of patients to health and usefulness before they become chronic invalids. But we are far from this desired goal. We are still treating most of our cases when the disease is advanced and far advanced, at a time when the psychical problems are real and serious. In early cases from nine months to a year and a half of active treatment will return most patients to lives of usefulness; in advanced cases the time is two or three times as long, and the result is less certain and less satisfactory. So it is evident that the psychic problems of the tuberculous patient vary much with the extent of the disease. Then, too, early cases as a rule have a minimum of ill feeling, while advanced cases often suffer greatly.

Tuberculous patients should always give attention to those things which will prevent their minds from degenerating during the time required for treatment. Reading, study, sewing, knitting, drawing, painting, making light mechanical toys, and other such diversions suited to the particular patient according to his condition will help him pass the time and at the same time bring him through his illness with a minimum loss of his powers of concentration and correlation. As soon as the patient is in a condition to exert himself, suitable exercise may be prescribed. I find walking the safest of all exercises for tuberculous patients. I try to have patients walk from one to five miles a day by the time they are through with their treatment. Thus they regain confidence while they have their

physician to guide them. Patients who follow out programs such as this are in a condition to assume light work soon after their active treatment is finished. Their morale is good. They know they can withstand exertion. They have not lost their powers of concentration. They may face return to home and business with a degree of security and confidence.

Not only does such a program bring the patient through his treatment but it relieves him of that awful defeatism which pervades the one who has not had the proper psychical aid and the proper physical rehabilitation. It too makes the treatment as pleasant as is possible for a regime which requires confinement and isolation from one's interests for long periods of time. That it shortens the time of treatment, too, is self-evident, for it relieves the patient of depressive and harmful emotions which hamper physiologic function; but best of all it preserves independence, self-respect, ambition, and fitness. Unfortunately, no matter how adequate the treatment, at times, patients are unable to assume their usual working obligations. They must either take a part-time job or wait longer and risk the danger of further deterioration while doing so. So attention should be given to rehabilitation and finding work such as these patients can do. It is important that they should become self-supporting as soon as possible after a successful result has been attained.

Our object is not alone to heal tuberculosis but to heal the patient who has it; to repair his psychologic injuries as well as those which have a physical basis; to restore him to the place where he can meet the problems of life without being hampered by a defeatism such as often follows chronic devastating disease. We should encourage him to gain proper control of himself and acquire a sane outlook on life so as to be able to approach his problems optimistically, in the easy way, with a confidence of being able to fill his position in life to the fullest capacity.

Non-Tuberculous Pulmonary Apical Disease

JOHN N. HAYES, M.D., F.A.C.P.
and
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A diagnosis of non-tuberculous pulmonary apical disease by its very title implies the rigid exclusion of tuberculosis. In a patient with a history of pleurisy with effusion, hemoptysis of one ounce or more and the presence of rales, after cough, in the upper one-third of the lung, we strongly suspect PULMONARY TUBERCULOSIS. The subsequent demonstration of parenchymal changes in the lung fields by means of the roentgen-ray, and acid fast bacilli in the patient's sputum enable one to make a definite diagnosis. The tuberculous patient may be asymptomatic but usually the onset of the disease may be classified as pleuritic, hemoptoic, insidious, or catarrhal, depending on the predominant symptom. An acute upper respiratory infection and so-called "grippe" from which the patient fails fully to recover may often be the beginning of pulmonary tuberculosis. The characteristic shadows in the x-ray film has a mottled, beaded appearance which may, or may not, have a hazy border, depending upon the predominating character of the reaction. In addition, cavitation may occur early in the disease and be identified as such in the stereoroentgenogram. These x-ray changes usually occur in the upper one-third of the lung fields. In the presence of cavitation the sputum should reveal acid fast bacilli on direct smear, culture, or guinea pig innoculation. A negative report in the presence of cavity should render the lesion suspect of being non-tuberculous. A negative Mantoux skin test, in the absence of high fever or terminal pulmonary tuberculosis, is not found in an active tuberculous lesion.

The characteristic onset of LOBAR PNEUMONIA with severe chest pain, chills, fever,

rusty sputum, dyspnea, and prostration, together with the physical signs of consolidation or, in early cases, distant or diminished bronchial breathing, should not cause difficulty. Typing of the sputum, isolation of the organism from the blood stream, and the typical diffuse shadow of lobar consolidation in the x-ray will clinch the diagnosis.

BRONCHOPNEUMONIA, especially in children, may be apical in location. Sputum is difficult to obtain and is not characteristic except that tubercle bacilli are not found. The chest plate may show patches of disease elsewhere than in the apex. Physical signs may be absent or may vary from time to time. A negative tuberculin skin test will be of value in these cases. In the adult the negative sputum is of the greatest aid in the diagnosis.

UNRESOLVED OR INTERSTITIAL PNEUMONIA may present a problem and is often tuberculous. Stringy shadows with an absence of mottling, broncho-pneumonic consolidation, negative sputa are aids in ruling out tuberculosis.

APICAL BRONCHIECTASIS (see Fig. 1, p. 13) may be associated with lung cysts, unresolved pneumonia, and fibrotic tuberculosis or may occur alone following some cicatricial process in the area. The constitutional symptoms are the usual ones of sepsis: chills, nightsweats, fatigue, loss of weight and strength, languor, anorexia, etc. Local symptoms are cough and purulent expectoration. The sputum is usually copious in amount (two to sixteen ounces) and may or may not have a rancid or fetid odor. Blood streaked sputum occurs in about one-half the cases and frank hemoptysis is not uncommon. The so-called "dry bronchiectasis" may occur without sputum but with repeated hemoptyses. Frequent examinations of the sputa fail to reveal pathogenic acid fast organisms, unless we are dealing with a tuberculous bronchiectasis. Spirochetes and fusiform bacilli are frequently found in the sputum. Physical examination of the chest is not of much aid — a few coarse rales may be heard — but the clubbing of fingers and toes is quite suggestive. The x-ray may reveal fibrosis and a suggestion of minute multilocular cavity formation. A striate appearance of the apex may afford a clue. The introduction of iodized oil into the bronchi leading to the suspected apex while the patient is lying on the affected side in a modified Trendelenburg position will afford a contrast bronchography which will reveal sacculation or dilatation of the bronchi in such cases.

LUNG ABSCESS (see Fig. 2, p. 13) may be preceded by pneumonia, septic embolic phenomena, trauma, operations on the upper respiratory tract, the aspiration of a foreign body (tooth, peanut, etc.) or vomitus and is frequently seen following exposure incident to an alcoholic debauch. The usual constitutional symptoms of sepsis are present. There may be a history of a sudden copious expectoration of foul sputum. Hemoptysis may occur if the abscess necrotizes a blood vessel. The sputum may be moderate in amount and foul smelling if infected with B. Coli or fusospirochetal organisms. No tubercle bacilli are found but a non-pathogenic acid fast organism may be seen on smears. Guinea pig innoculation is indicated in such cases. Elastic fibres are often found if there is extensive involvement. If the abscess has cavitated and is peripheral, the signs of cavitation may be heard. The x-ray may reveal a zone of decreased density with a surrounding area of haziness if the cavity has formed. In some cases, cavity formation may be as late as ten months, and before the cavity forms we may see a localised area of pneumonitis which may progress and recede at times. *THERE IS AN ABSENCE OF MOTTLING IN SUCH SHADOWS.* If the involved area excavates early and completely, the surrounding pneumonitis may be absent and to distinguish it from an early thin-walled tuberculous cavity is impossible without sputum studies.

PRIMARY TUMORS OF THE LUNG (see Fig. 3, p. 13) and pleura usually occur not earlier than the fifth decade of life. They are generally bronchogenic in origin but a few have been classified as pleural mesotheliomata. Loss of weight is the dominant constitutional symptom. Cough and expectoration almost daily, tinged or with frank blood; pain in the chest, persistent and severe; and dyspnea, if effusion be present, are the chief local symptoms. Pressure symptoms manifest by hoarseness, aphonia, or paralysis of the vocal cords, together with the objective find-

ing of diaphragmatic paralysis may be in evidence if the recurrent laryngeal or phrenic nerves are involved. There may be atelectasis if the bronchus becomes completely stenosed. No tubercle bacilli are found unless there is a combined lesion. Tumor cells have been identified, together with elastic fibres, in the sputum. The usual site of the x-ray lesion is near the root but it may be seen in the apex. The area of increased density has a stellate or irregular border suggestive of the invasive nature of the neoplasm. Metastatic tumors present a typical, sharply demarcated, border. The tumor may outgrow or occlude its blood supply and a central area of necrosis slough out to give the appearance of an apical tuberculous cavity. This may occur early. The dense homogeneous shadow of atelectasis may be seen in the upper lobes if the tumor occludes the bronchus. A massive effusion may obliterate the pulmonary markings. These effusions, when removed, are often bloody and microscopically tumor cells may be found. Bronchoscopy with biopsy will permit a definite diagnosis in most cases. Cervical or supraclavicular lymph nodes, if palpable, should be removed for microscopic study. Cutaneous nodules may occur that upon section will show tumor cells. Neoplasm must be kept in mind in all patients over fifty years of age who show a unilateral apical lesion with negative sputa. Extrapleural tumors such as lymphoma and aneurysm may cause pulmonary atelectasis by occlusion of a bronchus.

LUNG CYSTS (see Fig. 4, p. 13) more frequent in children, are usually congenital anomalies and are frequently associated with bronchiectasis. The patient may be symptom free or may have the symptoms of bronchiectasis. They may occur as an isolated apical rarefaction in the x-ray. Unless infected, they are thin-walled and without surrounding infiltration. A fluid level may be present. Acquired cysts may be due to parasites (echinococcus). Emphysematous blebs may resemble true cysts of the lungs. The sputum is negative for tubercle bacilli and is not characteristic. Contrast bronchography may be useful.

INTERLOBAR PLEURISY and EMPYEMA may be confused with apical tuberculosis. The condition develops from pneumonia, with a subsequent return of chills, fever, and other toxic manifestations. Suspended dullness over the fluid with resonance above and below it is characteristic. The x-ray film shows a sharply demarcated area of increased density, the lower border of which is almost horizontal. The lateral film localizes the effusion and a needle may reveal pus. There is generally no expectoration or the empyema may evacuate itself via a bronchus.

Rarer conditions to be kept in mind are atelectasis of the accessory lobe of the azygos vein, fungus infection, silicosis, and syphilis which may show a diffuse (fibroid) or conglommerate (gummatous) shadow on the x-ray but is rarely confined to the apex of the lung.

Tuberculosis Control*

Kentucky, along with the great majority of the other States in the Union, is showing an upward trend in mortality from tuberculosis. In 1936, deaths from this cause in the State totaled 2,065, as against 2,003 in 1935 — an increase of 62. This upward trend is just what students of tuberculosis expected and predicted as an inevitable result of years of acute economic depression. To them the surprise is that it did not come sooner.

The experience of the past two years has conclusively demonstrated:

1. That the effective control of tuberculosis demands as definite an epidemiological study of the disease as is required in the case of any other communicable disease.

2. That in any case-finding program, tuberculin testing, with X-raying of reactors, still constitutes the most dependable route.

3. That the private practitioner, who is tuberculosis conscious and applying modern methods of tuberculosis control, is the most important factor in solving the problem of finding active cases of tuberculosis and bringing them under treatment.

4. That it is highly essential that Parent-Teacher Associations, civic clubs, women's organizations and the citizenship generally interest themselves, actively and energetically, in the effort to insure that all school teachers, janitors and others whose duties bring them in close contact with school children, are free from infectious tuberculosis. No child is safe in school when and where one or more of the teachers or attendants are afflicted with tuberculosis in the infective stage.

5. That it is particularly important that high school groups all be tuberculin tested and that all reactors found among them be X-rayed. It is in such groups that the breaking down of the protective barriers against tuberculosis first manifests itself. Special attention should be given to high school students engaged in athletics.

6. That additional funds are needed to defray the expense of X-raying indigent reactors, and for the proper isolation of such as may be found to be spreaders of the disease. — John B. Floyd, M. D.

* Abstracted from the Kentucky Bulletin of the Department of Health, Vol. X., No. 5, December, 1937.

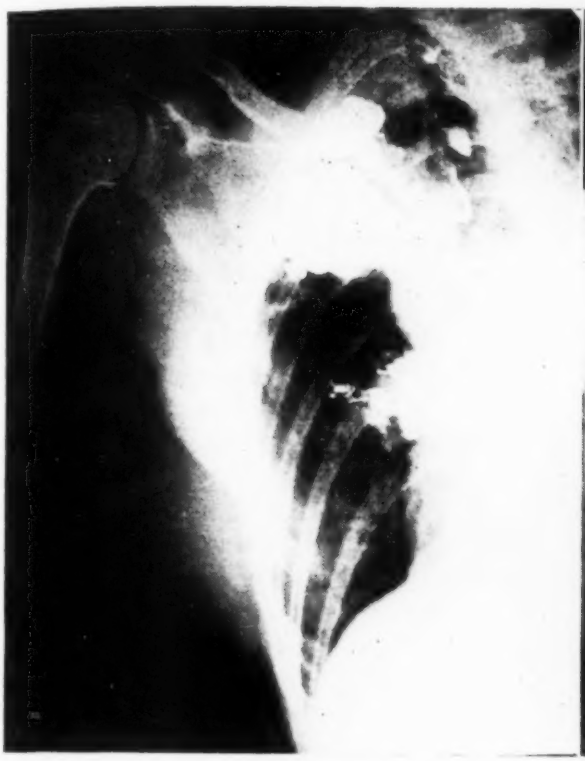

Fig. 1. Apical Bronchiectasis, Non-tuberculous.
(Courtesy of Dr. John Steidl, Trudeau Sanatorium, Trudeau, New York)

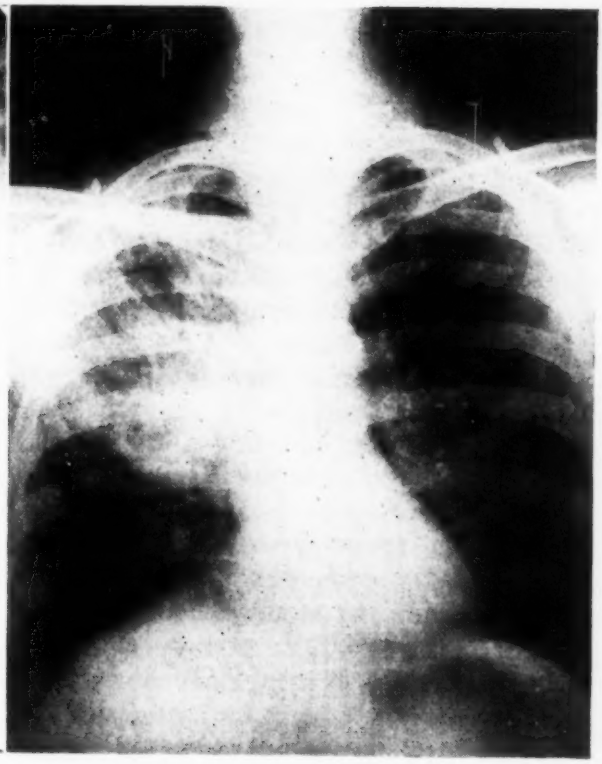

Fig. 2. Lung Abscess with Pulmonitis, Upper-third.
Death from cerebral abscess
Autopsy — no tuberculosis.

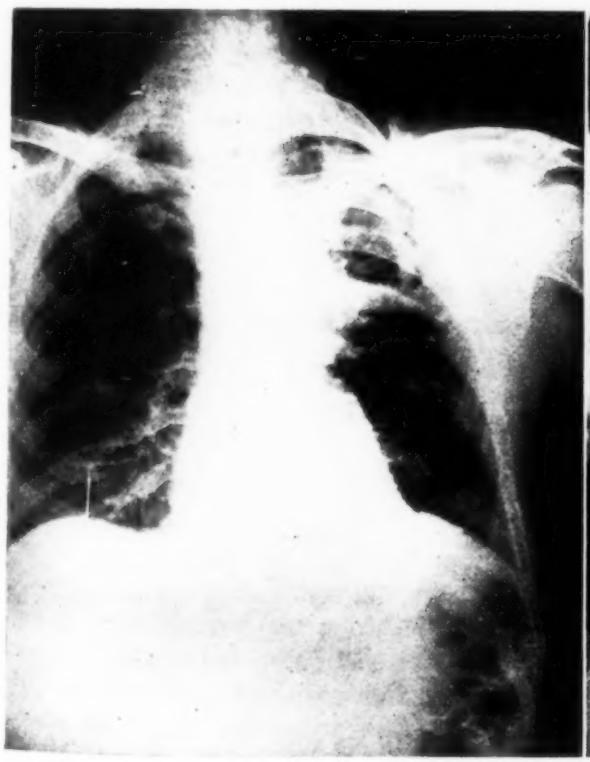

Fig. 3. Pulmonary Carcinoma with Cavity.
Proved at autopsy.

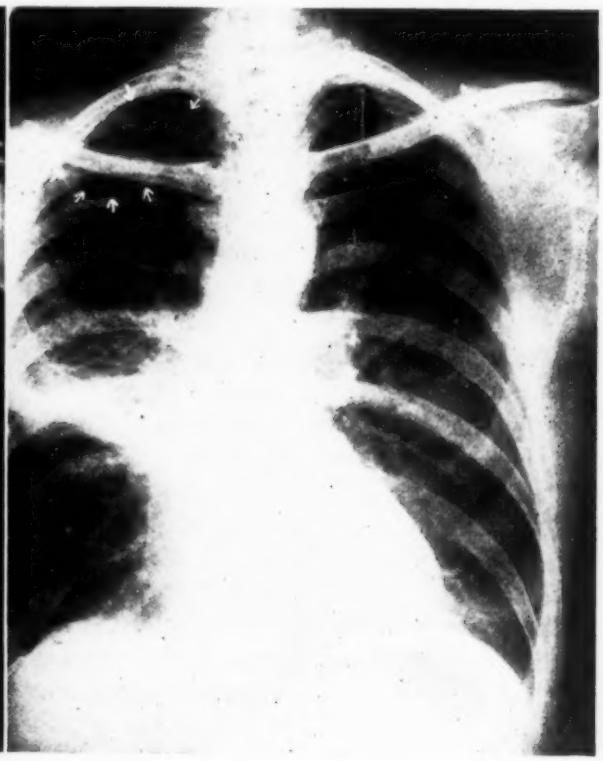

Fig. 4. Embolic Pneumonia, Lower Lobe, Quickly Resolved; Apical Cyst.
Death from cerebral-arterial accident.

Effusions Complicating Artificial Pneumothorax

Clinical Classification
Conservative Management, With The Use Of Oleo-Pneumothorax

CHARLES A. SMOLT, M.D.
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PLEURISY with effusion is the most common and potentially dangerous complication of pneumothorax therapy in tuberculosis. In a well-considered approach to the problem offered by the advent of such an effusion the disappearance of the fluid will not be made an end in itself. A lung is collapsed originally to provide favorable conditions for healing of tuberculous lesions within it. The real objects of treatment are; the preservation of these favorable conditions for healing, avoidance of damage due to systemic absorption of toxic material from the effusion, and satisfactory expansibility of the lung when the need for collapse has passed. If, through too much or too little treatment, the patient is rid of his effusion but left with an inexpansible lung or one which is being forcibly expanded by the contraction of massive adhesions his chances for recovery from tuberculosis have been seriously impaired.

While favorable effects, both immunologic and mechanical, have been ascribed to effusions in pneumothorax; a survey of the ultimate results of the long-continued presence of exudate in the pleural space will offer little comfort. Persistent effusions eventuate in a variety of ways, most of them tending to diminish the value of the pneumothorax. Those which remain serous lead to progressive thickening of both visceral and parietal pleurae, permanently enclosing the lung in a thick envelope, and promoting the growth of interpleural adhesions. Purulent exudates greatly increase the rate of pleural organization, are accompanied by fever and toxemia, and are prone to be complicated by fistulae and tuberculosis of extrathoracic organs.

The plan of treatment to be described has been employed in the tuberculosis service of Ventura County Hospital and in private practice for a period of five years. It has given acceptable results in a sufficient number of cases to warrant presentation. The procedures involved are not elaborate and have been frequently performed in patients' homes.

CLASSIFICATION

In considering management of pleuritic effusions in pneumothorax, it is useful to divide them into four classes:

I. *Small Intermittent Effusions.*

These appear slowly and give little symptomatic evidence of their presence, being discovered at routine fluoroscopy. The amount of exudate is seldom more than enough to cover the diaphragm. Usually disappearing in a few days or weeks, they may reappear from time to time during pneumothorax treatment without apparently affecting it.

II. *Clear Serofibrinous Effusions.*

Members of this class may appear at any time from the original introduction of air to several years afterward. The onset is that of a mild or severe acute infection, characterized by chills, fever, pain in the chest, nonproductive cough, anorexia, and more or less malaise. Air absorption from the pleura is reduced. Where the effusion accumulates rapidly a positive intrapleural pressure ensues and mediastinal shift may give rise to respiratory embarassment demanding relief. The fever and other symptoms of infection usually subside in from two to six weeks although fluid production may continue for months.

III. *Seropurulent and Secondarily Purulent Effusions.*

Although encountered most commonly as a secondary change in effusions belonging to Class II, seropurulent exudates may appear as such without a detectable clear serous stage. The symptoms are much the same as those of the preceding class except that complete defervescence is distinctly less common and the patients seldom regain the stage of convalescence enjoyed before the onset of

the pleurisy. Periodic exacerbations are attested to by increased fever, pain in the chest, and malaise. Slow deterioration in general condition, great thickening of the pleura, and an increased tendency to the development of extrathoracic tuberculosis are regularly seen.

IV. *Acute Purulent Effusions.*

This class is numerically small but of far greater immediate danger to the patient than any other. The pus may be of purely tuberculous origin or due to tubercle bacilli in combination with ordinary pus formers. Predisposing factors are; extensive tuberculosis near the surface of the lung, acute upper respiratory infections, influenza, and pneumonia. An occasional cause is the accidental wounding of the lung or a lung-containing adhesion by the needle. An especially severe type may follow the therapeutic collapse of a lung for non-tuberculous abscess. The onset is violent with chills, high fever, pain, dyspnea, sweating, and profound prostration. The most urgent symptoms may subside after a time and the case comes to resemble those in group III. In other instances however, the course is that of the most fulminating empyema resulting in early death.

MANAGEMENT

Class I.

The small intermittent effusions which outnumber all other types require little treatment. In some patients it is possible to reduce the frequency or volume of refills while the exudate is present, though more often the rate of air absorption is not much changed. At fluoroscopy the base of the lung near the diaphragm should be watched for a tendency to adhere. The use of the fluoroscope at regular short intervals is indispensable in caring for any patient having a pneumothorax effusion.

Class II.

Watchful waiting epitomizes the management of the early stages of pleurisy with a clear serofibrinous effusion.

Patients who have been out of bed should be returned to full bed rest for the duration of the rise in temperature and pulse rate. Acetyl salicylic acid or sodium salicylate dosage makes the patient more comfortable and appears to hasten defervescence. Pain in the chest or shoulder is often relieved by applying a firm binder around the abdomen and lower chest. Reassuring words to the patient and his family should not be neglected as it frequently appears to them that the situation is far worse than before pneumothorax was begun.

Reduction in the rate of pleural air absorption nearly always makes early refills unnecessary. Thoracentesis is best avoided until one of three situations arises: first, the fluoroscope shows that intrapleural pressure is too low and the lung tends to reexpand; second, symptoms and the fluoroscope point to so large a volume of fluid that respiration and circulation are embarassed; third, in the absence of pressure symptoms, the effusion becomes so abundant as to prevent adequate fluoroscopy. Adequate fluoroscopy demands a view of all the lung borders so that undue expansion of any portion is not overlooked. In the presence of more than 300 to 400 c.c. of fluid this means tilting the patient from side to side or having him lie upon his side in front of a vertical fluoroscope so that the fluid occupies a lateral position and reveals the base of the lung.

When the first situation arises, an ordinary air refill of sufficient size to meet the needs of lung collapse is made. A portion of the effusion may be aspirated through the same puncture, if necessary, to insure a better view of the lung. Unless a total collapse is indispensable to control of the intrapulmonary lesions it should not be maintained in the presence of any exudate as the prospects for reexpansion are always in jeopardy.

In the second situation, thoracentesis should be done and a sufficient amount of fluid removed to relieve dyspnea, allow the return of the mediastinum to its normal position, and permit a view of the lung borders. It is well to do this before excessive stretching of the mediastinum has occurred.

In the third case also, enough fluid is aspirated to bring the lung borders into fluoroscopic range. If needed to adjust the pressure properly an air refill completes the procedure.

If the effusion remains clear and no tendency toward pleural synthesis becomes evident, interference other than the procedures just described is not warranted for a period

of from three to five months. Fever attributable to the pleuritis commonly subsides within a few weeks and the patient regains the ground lost at the onset of the complication. In many cases the effusion is entirely absorbed within a few months and little trace of the pleurisy remains except that refills are not needed as frequently as before. In comparatively few patients are more than three tappings required to control the excessive production of fluid. However, the portion remaining after the last aspiration may show little tendency to be absorbed. The level seen under the fluoroscope rises and falls within narrow limits and, if all the fluid is aspirated, soon reappears at the same point. This state of affairs may continue indefinitely until the effusion becomes purulent or pleural thickening with adhesions changes the picture.

To avoid these contingencies, at the end of the period of waiting mentioned above—earlier if rapid fluid production persists—a partial oil replacement of the effusion is done. The oil mixture consists of 2 per cent gomenol in liquid petrolatum, warmed to body temperature. To test the patient's tolerance 2 c.c. of oil is introduced the first day followed by 10 to 20 c.c. upon the second. A few patients complain of the odor of the gomenol at first and may suffer some diminution in appetite for two or three days. Actual symptoms of intolerance are seldom encountered. After the preliminary doses, all but about 200 c.c. of the effusion is aspirated and is replaced with 300 c.c. of oil and enough air to adjust the intrapleural pressure. The case is then carried on as an oleo-pneumothorax. Some further exudation is to be expected and may be easily aspirated from below the oil as required. It is seldom of considerable volume and more than two aspirations between changes of oil are unusual. The oil may be removed and replaced every two or three months and after fluid formation has ceased may be discontinued. The favorable effect upon the pleura is further shown by an increase in the rate of air absorption.

More than 300 to 400 c.c. of oil are not employed since medication of the pleura is its only function and lung compression is still maintained by air. The disadvantages of a full oleo-thorax, such as the sensation of weight and the danger of fistula formation, are thus largely avoided.

Oil replacement should also precede an increase in intra-pleural pressure necessitated by formation of interpleural adhesions in the presence of serofibrinous effusions. While oleo-pneumothorax with positive pressure cannot be expected to stave off obliterative pleuritis permanently, it will often postpone the need for extrapleural collapse for long periods. The oil appears to exert a definite influence upon the formation and contraction of extensive adhesions.

Class III.

Management of seropurulent and secondarily purulent effusions differs but little in the first few weeks from that described for Class II, since the purulent character of the exudate cannot be established until thoracentesis is done. The true state of affairs may, however, be suspected before a tap is made because of higher temperature, more severe symptoms, and rapid accumulation of the effusion.

Every effort should be made toward preserving the patient's strength and insuring adequate nutrition. Pain in the chest may require strapping and codein or dilaudid. The low Fowler position is often preferable to a perfectly flat bed. Earlier interference is warranted in these cases than in those with clear effusions. Complete defervescence with satisfactory absorption of even a thin purulent exudate is seldom to be hoped for and few patients tolerate pus in the pleural space at all well. As soon as the symptoms of acute pleuritis have subsided and the daily fluctuations in temperature become about equal oil replacement should be considered.

Rapid complete replacements with high concentrations of gomenol are not necessary and frequently cause disagreeable reactions. It is best to begin by removing a small part of the effusion and introducing a few cubic centimeters of two or three per cent gomenol in petrolatum. After a few days the operation is repeated on a larger scale and a third sitting should empty the chest of exudate and leave in its place about 400 c.c. of oil containing 4 per cent of gomenol. This often results in a great improvement in the patient's condition and, in favorable cases, no higher

concentration of gomenol need be used. Reaccumulation of exudate is easily detected at fluoroscopy since the less radiopaque oil floats upon it.

The volume of exudate and the patient's temperature dictate the intervals at which tapping need be done and each time the chest may be emptied of fluid and oil followed by replacement with fresh oil. When lower concentrations of gomenol fail to produce the desired improvement, solutions may be employed containing from 10 to 12 per cent. It is best to increase the concentration gradually. As the rate at which the exudate forms decreases a change in its character toward a serous or serosanguinous type is noted and an increase in air absorption evidences better condition of the pleura. After several months to a year or more, the use of oil may be discontinued altogether.

In doing oil replacements I do not perform a preliminary lavage of the pleural space since it adds appreciably to the length of the procedure and the chance of shock. If difficulty is met with in aspiration because of thick exudate or fibrinous flakes, a few ounces of normal saline solution containing powdered caroid is instilled and allowed to remain for a few minutes. This proteolytic enzyme will render a thin easily aspirated fluid and does not seem to have any unfavorable effect.

Class IV.

Successful management of an empyma of this class taxes the resources of the pneumothorax therapist and his consultant surgeon to the utmost. The patient is dangerously ill almost from the onset and because of his collapsed lung is without means of localizing the infection.

Treatment during the acute febrile period is directed largely toward supporting the patient in his struggle to survive a severe and widespread infection. Nutritional and fluid requirements often need be met by intravenous saline and glucose solutions. Blood transfusion may be imperative and should be used early and repeated at need. Pain in the chest with its resultant dyspnea is often greatly relieved by oxygen inhalation and the application of a binder. Short wave diathermy applied through the affected half of the chest gives much subjective relief.

Thoracentesis should not be done within the first week unless a high intrapleural pressure becomes evident. Thereafter however, if large quantities of pus are present, frequent aspiration will have to be done or a catheter introduced into the chest and closed drainage instituted. If sufficient improvement in the patient's condition is obtained, an attempt to disinfect the pleura with gomenol in oil should be made and in some cases the pneumothorax is saved thereby. Lavage with warm normal saline solution with or without the addition of an antiseptic assists in the removal of the pus but is not often of great assistance in overcoming the infection. Rib resection may have to be resorted to if these measures fail to give adequate drainage because of "pocketing" and thick pus.

In a large percentage of patients with acute purulent effusions hope for continuance of a useful pneumothorax must be abandoned and the objective changed toward preparing the patient for some form of extrapleural collapse. This is beyond the scope of this paper.

SUMMARY

1. A working classification of effusions complicating artificial pneumothorax is presented.

2. In clear serofibrinous and in seropurulent effusions, interference for definite reasons only, with conversion to oleo-pneumothorax, offers a widely applicable means of preserving useful lung collapse.

Diagnostic Features of Silicosis and Silico-Tuberculosis

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IT IS generally recognized today that silicosis is the most common and, from the standpoint of medical interest, the most important of the hazardous forms of pneumoconiosis. It is also well known that the hazards of silicosis in its effect upon the death rate in pulmonary tuberculosis is very high. Silicosis has the tendency to produce increased susceptibility to pulmonary infections and particularly to tuberculosis. Latent tuberculous foci or childhood type of tuberculosis may become reactivated by silicotic changes or a preexisting silicotic process may become complicated by a superimposed tuberculous condition through external infection with tubercle bacilli.

Pathological Changes in Silicosis. Silicosis develops by breathing air which contains very fine particles of silicon dioxide dust, also known as free silica or quartz. The microscopic silica dust which is capable of producing pathological changes in the parenchyma of the lungs is only two to five microns in diameter; the dust particles which are more than ten microns in size are usually harmless. The inhaled dust, when lodged in the alveoli of the lungs, is first carried into the lymphatic circulation by the phagocytes. Thus, the dust becomes deposited in the tracheobronchial lymph glands producing there pathological changes first. But when the inhalation of dust continues, supersaturating the lymphatic system, then as the next step, the phagocytes deposit their dust particles in the cells of the lungs. The presence of foreign particles in the lung tissue stimulates a protective reaction resulting in the formation of thick fibrous tissue walls around the dust particles. In this manner silicotic nodules are formed. They are from one to six mm. in diameter. The nodules progressively increase in size. When there is a conglomeration of nodules localized in a certain portion of the lungs, there occurs a coalescence of the nodules into larger silicotic areas. Thus, silicosis is a progressive disease. Nodulation and formation of new fibrous tissue continues in the victim even after the exposure to dust has ceased. The damage produced in the lungs is permanent. It was formerly believed that the pathological changes in the parenchyma of the lungs was due to the mechanical action of the sharp dust particles. At present most authorities agree that the chemical action of silicon dioxide stimulates fibrotic changes. Silicon dioxide, which is slightly soluble in the alkaline body fluids, produces toxins which are supposed to initiate fibrosis and nodular formation. The process is always bilateral, occuring simultaneously in both lungs. The length of the process varies from several months to twenty years.

Where silicosis and tuberculosis coexist, the pathology in the lungs corresponds to the changes produced by both diseases. Nodulations and connective tissue fibrosis are scattered within and about tuberculous infiltrations, forming massive areas of chronic disease. Chronic adhesive pleurisy is a frequent complication. The occurrence of cavities and caseation is infrequent. Again, some other types may present a somewhat different picture, characterized mainly by perinodular tuberculous processes. The silicotic nodules may become caseous, being surrounded by exudative tuberculous infiltrations. In silicosis, complicated by tuberculosis, it may be said generally, that the pathological changes correspond to the type of tuberculosis present.

General Diagnostic Considerations. There are no specific signs or symptoms in silicosis or silico-tuberculosis that are not found in other pulmonary diseases. Shortness of breath and disability are the main symptoms. For diagnostic purposes the disease is sometimes divided into three stages. In the first stage the symptoms are not very marked, except possibly for some shortness of breath. There is no disability at first. As the disease progresses, the respiration becomes gradually

more and more affected. Then the most characteristic symptom is marked dyspnea. The breathing is labored and disability also sets in gradually. But later, a chronic cough may develop; the lung capacity for breathing decreases; weakness progresses and finally complete disability occurs. In this final stage, the victim is likely to develop tuberculosis which becomes fatal. Deaths from uncomplicated, single silicosis are infrequent. The physical signs are of no help in differentiating the disease from other pulmonary conditions which might be present. But a physical examination is of greatest importance because, in the first place it leads to the determination of the presence of some pathology in the lungs, and then it aids in determining the presence of some type of infection complicating silicosis. In difficult borderline cases, the physical examination proves especially valuable. Again, a physical examination alone is absolutely insufficient for establishing the diagnosis. For this purpose, a most carefully obtained history and an x-ray examination become absolutely indispensable. For the diagnosis of tuberculosis superimposing silicosis, sputum examinations are required.

History. In determining the presence of silicosis, a good history is the most essential factor. The history must be very complete, covering not only the type of work done, but also the length of time spent in a certain occupation. In taking the history it is of great importance to ask the individual about all occupations in a chronological order, from the day the victim has left school and become involved in gainful work until the onset of the present disease in question. The purpose of this procedure is to discover whether an individual who recently had a dustless occupation might not have been exposed to inhalation of silica dust sometime during his life. Silicosis is a progressive disease and it may be traced to a dusty occupation many years back in the past. The fact that somebody, discontinued many years ago a dusty occupation, does not eliminate silicosis. The disease is permanent and the progress of the disease continues even when exposure to silica dust has been discontinued for many years. On the other hand, not being directly involved in a dusty occupation does not eliminate the chances of acquiring silicosis. Working close to a place from which fine silica dust may be blown in many directions for a distance of many feet, creates a potential environment of exposure. Besides mining and quarrying there are many other industries where silica hazards exist, such as: foundries, potteries, glass factories, paint factories using silica, and occupations such as grinding, sand-blasting, etc. The concentration of silica dust varies in different industries. The concentration of silica dust determines the extent of pathology and the length of time necessary to produce silicotic changes in the lungs. The higher the concentration of the silica dust the less time will be required to produce bad effects, and the extent of pathological changes will be greater. The length of exposure required varies, therefore, with the different occupations in various industries. Among foundry workers the risk of acquiring silicosis is comparatively lower than in many other industries and the time of exposure, therefore, would be longer. But occupations which involve a high concentration of fine silica dust cause a rapid progress of the disease, and silicosis may occur in as short a time as a few months. Therefore, the details of the type of occupation in a certain industry and the length of time of exposure must be necessarily included in the history.

X-ray diagnosis. Equally important as the history is the x-ray examination. It is superfluous to say that the x-ray plates must be taken by an expert and read by an experienced radiologist. For the purpose of diagnosing silicosis or silico-tuberculosis, the best possible films are required, showing the minutest structural changes in the lungs. Improper exposures may not reveal the most desirable and most characteristic features, thus increasing the difficulties in obtaining a definite diagnosis. The silicotic nodulations on the x-ray plate appear as well-defined shadows about 6 mm. or less in size of uniform density. They are distributed bilaterally, with somewhat lesser distribution in the lower portions of the lungs. When there is coalescence of nodules, localized in certain areas of the lungs, there appear conglomerate shadows on the x-ray film. Silicosis may simulate many other lung conditions such

as miliary tuberculosis, fungi infections, etc. By taking a series of x-ray exposures usually other diseases may be eliminated. In order to obtain a better idea of the distribution of silicotic nodulations, stereoscopic plates are recommended. Some authorities believe that stereoscopic plates and a series of x-ray exposures are essential requirements for diagnosis. When silicosis is complicated with tuberculosis, the reading of x-ray films becomes essentially more complicated. Cavities may be seen only in far-advanced cases.

Sputum examinations. In order to be able to determine whether silicosis is complicated with some type of lung infection and in particular with tuberculosis, repeated sputum examinations are of primary importance. Routine sputum examinations should be done in every case. When the sputum is negative for tuberculosis, animal inoculations of the material will sometimes prove of great help in establishing the diagnosis.

Conclusion

1. Silicosis is a permanent and progressive disease, characterized by nodular formations and fibrosis in the lungs.

2. Silicosis has the tendency to increase susceptibility to pulmonary tuberculosis.

3. Shortness of breath on exertion, cough and lowered lung capacity are the common symptoms in silicosis.

4. The physical examination is extremely valuable but alone is insufficient for establishing the diagnosis.

5. The history is of primary importance, but it must be complete and exhaustive with special reference to the type of occupation in the silica dust environment and the length of time of exposure.

6. Perfect x-ray plates are indispensable. They must be interpreted by an expert radiologist. The distribution of silicotic nodules is bilateral.

7. Routine sputum examinations are required.

Lipiodol Bronchography*

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FROM the time that the roentgen ray has been used as a diagnostic agent in studying physiological action and pathological changes, physicians and research workers have increasingly sought suitable contrast media.

At the present time, we have our contrast meals for the gastro-intestinal tract, enhanced in recent years by the added ability to visualize the actual mucosal pattern of the tract; likewise, in the urinary tract, retrograde pyelography and intravenous urography and cystography; cholecystography for studying function and lesions of the gall bladder; lipiodol installation of the paranasal sinuses; air as a contrast medium in ventriculography and encephalography, pneumoperitoneum, double contrast enemata study of the colon; arteriography; myelography visualization of fistulous tracts, as well as others.

Roentgenologic contrast study of pathological changes in the lungs and bronchial tree was advocated as early as 1918, when Chevalier Jackson[1] attempted bronchoscopic insufflation of bismuth and barium powders, while H. L. Lynah[2], in 1920, employed a mixture of bismuth and pure oil for the localization of an abscess cavity.

The first experiments with lipiodol, as a diagnostic agent, were made by J. A. Sicard[3], and papers published on the subject in 1922. Subsequent to this, his assistant, J. Forestier[4], in conjunction with Leroux, injected lipiodol in the human trachea. Since that time, others have come to use it, although we feel that only the surface of its diagnostic possibilities in pulmonary lesions, has been touched.

Lipiodol application to the bronchial tree has a two-fold purpose: diagnostic and therapeutic.

Diagnostic indications are:

*Delivered before the meeting of the American College of Chest Physicians at Atlantic City, June 7th, 1937.

1. To determine the source of coughed up purulent non-tuberculous sputum.
2. To determine the point of blockage in the various types of atelectasis.
3. To determine the open or closed condition of abscesses or tuberculous cavities.
4. To demonstrate the amount of constriction and the location of the constriction in bronchial stenosis and spasm, such as asthma.

Therapeutic indications:

Lipiodol is useful in the treatment of the following conditions:

1. All forms of asthma.
2. Bronchiectasis.
3. Chronic retentive bronchitis.

It is probable that further uses for lipiodol will be demonstrated as time goes on.

It is noted that irrespective of the cause or duration of asthma, the instillation of lipiodol into the bronchial tree is promptly followed by the expectoration of opaque pus with a relaxation of the spasm. The frequency of treatments necessary to maintain improvement varies in different cases, but taking into consideration that mild hay fever often causes the severest asthma and vice versa, one is led to suspect that the asthma in any case is dependent to some extent upon the emptying ability of the bronchial tree in the individual case and upon imprisonment of purulent secretion below the point of bronchiolar constriction.

Lipiodol is a poppy seed oil containing 40 per cent iodine. It is opaque to x-rays. The iodine is so combined with the oil, that in the bronchial tree the iodine is liberated so slowly that it is non-irritating. The iodine content adds so much weight to the oil that gravity carries it to the bottom of cavity or bronchus, mechanically floating any contained pus or secretion to a level where it can be coughed up.

The patient's throat is sprayed with a 10 per cent cocaine solution while the tongue is held far forward, so that no cocaine enters the esophagus. Cocaine, even in a 10 per cent solution, is not absorbed from the bronchial tree or pharynx sufficiently to give symptoms. After cocainization, and with the tongue held in the same position, lipiodol is applied to the bronchial tree by dropping it on the base of the tongue during respiration. Following the installation of the lipiodol routine anteroposterior and lateral roentgenograms, are made.

Preliminary fluroscopic study may be made at the discretion of the clinician and roentgenologist. It is not a necessity. We would, however, like to stress the importance of the lateral bronchogram. By reason of the anatomical overlap of the lobes of the lungs, this projection will help to separate the overlapping shadows of the bronchial tree. The various oblique views are made according to necessity, after a wet film survey is made of the first two roentgenograms. One reason for not routinely fluoroscoping is our desire to get the bronchograms before the patient coughs.

Technique is a bit different from the ordinary roentgenograms for lung detail. Bronchography being a contrast study, we prefer to sacrifice the finer lung markings for blacks and whites.

The time of excretion of lipiodol is a moot question that still must be settled by research. In some cases the lungs are clear in twenty-four hours, while in others the lipiodol may remain for months, and in one personal experience, a cluster of small shadows were observed after a period of two years.

Ballon and Ballon[5], claim that the oil is eliminated more quickly from diseased than healthy areas. Looking at this question from different aspects, one might argue from several angles as to its reliability. If we start from the premise of the ciliated epithelium and its action, similar to that of the paranasal air cells, then we would expect oil to remove more quickly from the normal areas. Proetz, in his study of these sinuses, set seventy-two hours as the dividing line between normal and pathological. However, if one compares these to inflamed and irritable mucosa of the gastro-intestinal tract, then the reverse effect, or that claimed by Ballon and Ballon, would hold good. Cough impulse must also be taken into consideration.

In the trachea and larger bronchi there will be developed a normal and abnormal mucosal pattern, just as has been developed in the gastro-intestinal tract and especially similar to the roentgenographic picture of the double contrast study in the colon.

Most of our observations have been made upon bronchiectatic lesions. We have come

to recognize three types, or three stages, of the same lesion.

First; Tubular: The outline of the bronchial aborisations show a nodular outline instead of the normal smooth appearance, with increase in bronchial diameter.

Second; Fusiform: The typical clubbed finger picture is observed. Depending upon the stage, the contrast material may or may not enter the alveoli; if there is a distinct filling defect, localized bronchiectatic atelectasis is indicated.

Third; Saccular: There occurs the rounded grape-like outlines of lipiodol, either completely filled or showing a fluid level.

Naturally, by reason of dependency, the bases are more liable to bronchiectatic lesions than elsewhere, and the right side, more than the left. The upper branches of the bronchial tree are not immune, however. Like any lesion elsewhere, one must be on the lookout for the early changes and this requires a careful scrutiny of the various branches of the bronchial tree.

Bronchography has added a very valuable adjunct in the diagnosis of bronchiectasis for by no other means can it be definitely diagnosed, particularly early enough for therapy to be of any real effect. Likewise, the contrast study permits one to serially study the progress of a patient and we do see striking results, both symptomatically as well as structurally.

One is many times surprised at the displacement of right and left main stem branches. This finding is invariably the result of collapse of a lobe with emphysematous distention of the superimposed or underlying lobe. The resulting x-ray pictures of the bronchus, resembles a steerhorn.

In some cases the tuberculous cavities fill readily, and bronchography offers another means of differentiating cavities, localized pneumothoraces and pleural blebs or rings.

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Aphorisms Concerning Diseases of The Chest *

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EMPYEMA of the thorax should not be drained early. The first impulse, when pus forms, is to get rid of it at once. But in the case of empyema, this means operation, while the patient is still acutely ill with pneumonia, and it also means the induction of a massive pneumothorax, while the patient is still acutely ill. Closure in such a case is usually very difficult, because of the extensive pneumothorax. The proper treatment is early aspiration, and if necessary, the empyema may be drained later. By so doing, we can choose the time of operation and avoid operating on an acutely ill patient. If the pus is kept aspirated to a low level, adhesions will form low down; massive pneumothorax will not result from the drainage operation, and closure will be relatively easy. Empyema complicating artificial pneumothorax seldom demands drainage.

Pleurisy with effusion is nearly always tuberculous. The extent of the tuberculosis is usually slight, and this fact caused the etiological significance of pleural effusions to be overlooked until comparatively recent years. It is probable that the pleural effusion forms as a result of the irritation of allergic cells in the pleura by slight tuberculous infection. Examination of the fluid for tubercle bacilli by the smear method is usually negative, and even guinea pig inoculation has been known to show negative results in cases that later proved to be tuberculous. If the patient has an idiopathic pleurisy with effusion, it is best to tell him that he probably has tuberculosis and warn him as to the dangers.

Onset of Tuberculosis. Two types of onset

*Reprinted from Kansas City Medical Journal.

are common in pulmonary tuberculosis.

A. The Insidious Onset.

The insidious onset is characterized by a triad of symptoms. Significant symptoms are cough and expectoration, fever and tachycardia, loss of weight and strength. When a patient has these symptoms for three months, or more, the diagnosis is tuberculosis, until it is disproven.

B. The Sudden Onset.

The sudden onset is characterized by what appears to be an attack of influenza or influenza with pneumonia. If such a case fails to get well within a short time and continues to run fever, the chest should be carefully studied with x-ray plates to see if tuberculosis is present. Persistent search in the sputum for tubercle bacilli may clear the diagnosis.

Chronic Pneumonia, usually called "unresolved pneumonia" is due usually to either empyema or tuberculosis. Every case of chronic pneumonia should be carefully investigated to determine why the toxemia continues.

Hemoptysis of a definite quantity, say one dram or more of bright red blood, followed for hours or days by blood-streaked sputum, is usually due to tuberculosis. If a patient has a definite hemoptysis which cannot be otherwise explained, he should be regarded as having pulmonary tuberculosis, even though physical examination of the chest and x-ray plates are negative. Hemoptysis is occasionally due to bronchiectasis.

Sputum Examination. One negative sputum is worth very little in disproving a diagnosis of tuberculosis. If the case is a suspicious one, persistent search should be made through a great number of specimens, and even then bacilli may not be found, even though the correct diagnosis is tuberculosis.

Artificial Pneumothorax. Almost every case of tuberculosis which comes to a fatal conclusion passes through a stage when artificial pneumothorax should be applied. If we studied our cases carefully, from their inception, pneumothorax would be used much more widely, and many more lives would be saved.

Rest is all important in the treatment of tuberculosis. I am accustomed to telling my patients that the first principle of treatment is rest. The second principle of treatment is *Rest,* and the third principle of treatment is *R-e-s-t.*

Asthma. When an attack of asthma is quickly and completely relieved by the injection of adrenalin, it is usually due to allergy. In this way, adrenalin becomes a very good diagnostic agent, for it aids in separating the allergic from the mechanical and reflex asthmas. If adrenalin consistently fails to relieve the attack of asthma, the asthma is probably not due to allergy.

Bronchiectasis. I believe that most cases of bronchiectasis have two etiological factors, (1) a congenital weakness of the bronchial wall, and (2) retention of secretions as a result of infection. The latter factor is one which may be attacked with some success. Infection in the lower bronchial tree results in irritation. Irritation produces hypersecretion. The hypersecretion leads to retention, and the retention results in further infection. Thereby, a vicious circle is produced which results ultimately in distention of the bronchial tree. The best way to attack this vicious circle is to prevent retention. In draining the bronchial tree, three cardinal principles are involved. Compare the bronchial tree with a bottle. In order to empty it, you must get the cork out, thin the contents and invert the bottle. This means increase the caliber of the bronchial tubes, liquify the bronchial content and use postural drainage.

Total Left Pneumonectomy for Primary Bronchogenic Carcinoma**

Case Report*

THOMAS J. KINSELLA, M.D.

Oak Terrace, Minnesota

I AM very sorry that my patient was unable to appear before you this evening but a slight indisposition, unrelated to the present illness, prevented him from making the automobile trip to the city for this meeting.

This man, sixty-one years of age, a hotel keeper and former railroad conductor, first consulted me on June 21, 1937, referred by Dr. E. E. Carpenter of Superior, Wisconsin, because of cough, fever, and loss of weight. He had apparently been perfectly well until the middle of December, 1936, when he developed an acute tonsillitis followed by an arthritis of the right great toe which persisted for two weeks. With this tonsilitis he had a fever to 101 degrees and cough and expectoration of colorless mucoid material amounting to one-half ounce in twenty-four hours. When the acute process subsided, cough and expectoration of clear mucous and slight dyspnea without wheezing persisted. His chest was negative to physical and fluroscopic examination on January 20, 1937. The cough and expectoration without blood or pus continued with increasing loss of strength and weight which by April totaled twenty-three pounds. X-ray examination at this time revealed an infiltration about the hilum of the left lung. After a period of four weeks treatment at a rest home, he was transferred to the Middle River Sanatorium at Superior, where he remained until June 20, 1937. The cough expectoration and weight loss continued in spite of bed rest until he had lost a total of thirty-three pounds in a period of six months. Repeated sputum examinations by smear and guinea pig innoculations were negative for mycobacterium tuberculosis.

My examination revealed a large, well developed man presenting evidence of marked weight loss. He was troubled by a frequent slight cough with expectoration of clear mucoid material. Physical examination revealed obstructive emphysema of the left upper lobe and roentgen examination showed an increased infiltration in the left lung as compared with one month previously. Bronchoscopic examination by Dr. L. R. Boies revealed mucoid secretion coming from the upper lobe bronchus but no tumor mass. A diagnosis of obstruction of the left upper lobe bronchus probably due to malignant tumor without evidence of metastasis was made and exploration recommended. A complete x-ray study of the gastro-intestinal tract revealed no evidence of new growth.

The patient was admitted to Northwestern Hospital on July 7, 1937, and left pneumothorax induced as a preliminary to thoractomy. This was continued for a period of ten days and a 50 per cent collapse of the lung obtained. Bronchoscopic examination was repeated in the hope of visualizing a tumor in the upper lobe bronchus and obtaining a biopsy to establish the diagnosis but again there was no mass to be seen. The obstruction of the upper lobe bronchus persisted and the patient continued to run a mild septic type of temperature from secondary infection in the obstructive lobe.

On July 20, 1937, under intra-tracheal cyclopropane anesthesia, administered by Dr. Ralph T. Knight with the patient lying on his back, the left pleural cavity was opened by long transverse incision through the third interspace anteriorly without resection of the rib but with a parasternal section of the third and fourth cartilages and the incision widened by means of a rib spreader. Extensive adhesions encountered anteriorly and medially were divided and a total left pneumonectomy performed according to the Rienhoff technic. The mediastinal pleura was opened and the left pulmonary artery and veins doubly ligated and sectioned in se-

* Reprinted from Minnesota Medicine, Vol. 20—No. XII. — December 1937.

** Read before the meeting of the Minneapolis Surgical Society, October 7, 1937.

quence. The left main bronchus was divided, without crushing, about fifteen millimeters from the carina and after section of the cartilages at two points was closed with interrupted silk sutures. The bronchial stump was allowed to retract into the mediastinum and the mediastinal pleura closed over it. The chest incision was then closed tightly without drainage. Intravenous fluid and blood were given during the operation as a precautionary measure but the procedure as a whole was well tolerated and the patient left the operating table with his skin warm, dry and pink and a pulse of 84, respiration of 20, and blood pressure, 124/80. The excised lung revealed a squamous cell carcinoma, which almost completely obliterated the upper lobe bronchus without any projection into the stem bronchus. There was considerable secondary pneumonitis and bronchiectasis distal to the tumor mass. The patient's postoperative condition was relatively uneventful. He remained in an oxygen for twenty-four hours and was then removed as there was no evidence of anoxemia. The left pleural cavity rapidly filled with fluid which was not disturbed as no pressure symptoms developed. Subsequent aspiration two weeks after the operation revealed a straw colored clear fluid with a cell count of 200 per cubic millimeter. The wound healed by primary intention, the patient was permitted to get out of bed at the end of two weeks and walked out of the hospital at the end of the fifth week in good condition. His subsequent course has been uneventful. A detailed report will be published at a later date. To our knowledge, this is the first successful total pneumonectomy performed in the state of Minnesota.

ORGANIZATION NEWS

To our Fellows of the American College of Chest Physicians: Best Wishes For A Happy New Year.

Remit your dues for 1938 promptly; make your check payable to the American College of Chest Physicians, and mail to Dr. R. B. Homan, Jr., Secretary-Treasurer, P. O. Box 1069, El Paso, Texas.

Notify this office at once if there is a change of address, or if your present listing in the 1937 Pneumothorax Directory is incorrect. This information is necessary to assure correct listing in the *1938* Pneumothorax Directory.

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